Cannabinoid

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Cannabinoids (pronounced /ˈkænəbɪnɔɪdz/, *KA-nə-bi-noydz*) are a group of terpenophenolic compounds present in Cannabis (*Cannabis sativa* L) and which occur naturally in the nervous and immune systems of animals. The broader definition of cannabinoids refers to a group of substances that are structurally related to tetrahydrocannabinol (THC) or that bind to cannabinoid receptors. The chemical definition encompasses a variety of distinct chemical classes: the classical cannabinoids structurally related to THC, the nonclassical cannabinoids, the aminoalkylindoles, the eicosanoids related to the endocannabinoids, 1,5-diarylpyrazoles, quinolines and arylsulphonamides and additional compounds that do not fall into these standard classes but bind to cannabinoid receptors.[1] The term cannabinoids also refers to a unique group of secondary metabolites found in the cannabis plant, which are responsible for the plant's peculiar pharmacological effects. Currently, there are three general types of cannabinoids: *phytocannabinoids* occur uniquely in the cannabis plant; *endogenous cannabinoids* are produced in the bodies of humans and other animals; and *synthetic cannabinoids* are similar compounds produced in a laboratory.

Contents

Cannabinoid receptors

Before the 1980s, it was often speculated that cannabinoids produced their physiological and behavioral effects via nonspecific interaction with cell membranes, instead of interacting with specific membrane-bound receptors. The discovery of the first cannabinoid receptors in the 1980s helped to resolve this debate. These receptors are common in animals, and have been found in mammals, birds, fish, and reptiles. There are currently two known types of cannabinoid receptors, termed CB1 and CB2, with mounting evidence of more[2].

- CB1 receptors are found primarily in the brain, specifically in the basal ganglia and in the limbic system, including the hippocampus. They are also found in the cerebellum and in both male and female reproductive systems. CB1 receptors are essentially absent in the medulla oblongata, the part of the brain stem that is responsible for respiratory and cardiovascular functions. Thus, there is not a risk of respiratory or cardiovascular failure as there is with many other drugs. CB1 receptors appear to be responsible for the euphoric and anticonvulsive effects of cannabis.

- CB2 receptors are almost exclusively found in the immune system, with the greatest density in the spleen. While generally found only in the peripheral nervous system, a report does indicate that CB2 is expressed by a subpopulation of microglia in the human cerebellum [3]. CB2 receptors appear to be responsible for the anti-inflammatory and possibly other therapeutic effects of cannabis.

Phytocannabinoids

Phytocannabinoids, also called *natural cannabinoids*, *herbal cannabinoids*, and *classical cannabinoids*, are only known to occur naturally in significant quantity in the cannabis plant, and are concentrated in a viscous resin that is produced in glandular structures known as trichomes. In addition to cannabinoids, the resin is rich in terpenes, which are largely responsible for the odour of the cannabis plant.

Phytocannabinoids are nearly insoluble in water but are soluble in lipids, alcohols, and other non-polar organic solvents. However, as phenols they form more water-soluble phenolate salts under strongly alkaline conditions.

All natural cannabinoids are derived from their respective 2-carboxylic acids (2-COOH) by decarboxylation (catalyzed by heat, light, or alkaline conditions).

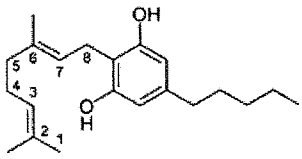
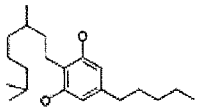
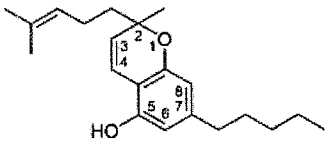
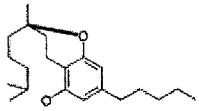
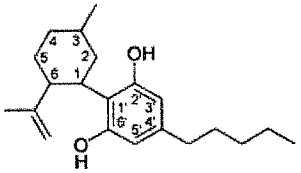
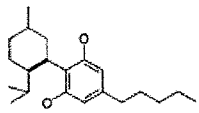
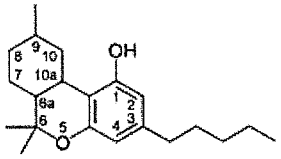
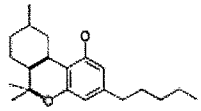
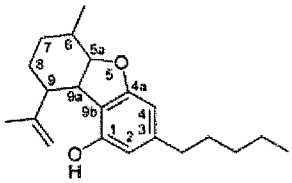
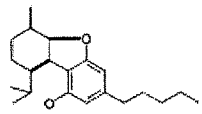
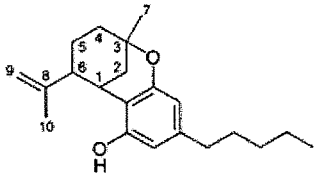
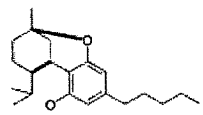
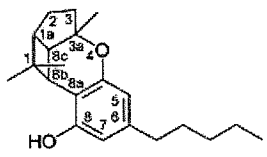
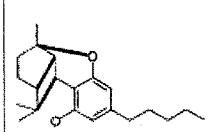
Types

At least 66 cannabinoids have been isolated from the cannabis plant[4] To the right the main classes of natural cannabinoids are shown. All classes derive from cannabigerol-type compounds and differ mainly in the way this precursor is cyclized.

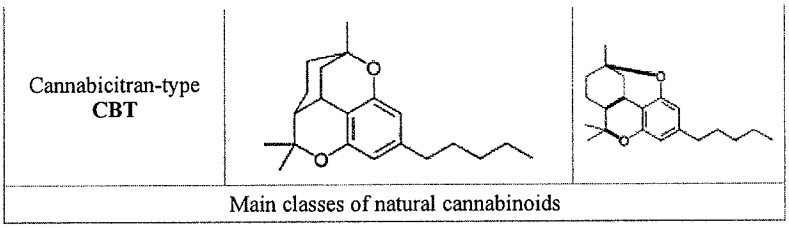
Tetrahydrocannabinol (THC), cannabidiol (CBD) and cannabinol (CBN) are the most prevalent natural cannabinoids and have received the most study. Other common cannabinoids are listed below:

- CBG Cannabigerol
- CBC Cannabichromene
- CBL Cannabicyclol
- CBV Cannabivarin
- THCV Tetrahydrocannabivarin
- CBDV Cannabidivarin
- CBCV Cannabichromevarin
- CBGV Cannabigerovarin
- CBGM Cannabigerol Monoethyl Ether

Tetrahydrocannabinol

Type	Skeleton	Cyclization
Cannabigerol-type **CBG**		
Cannabichromene-type **CBC**		
Cannabidiol-type **CBD**		
Tetrahydrocannabinol- and Cannabinol-type **THC, CBN**		
Cannabielsoin-type **CBE**		
iso-Tetrahydrocannabinol-type ***iso*-THC**		
Cannabicyclol-type **CBL**		

Tetrahydrocannabinol (THC) is the primary psychoactive component of the plant. Medically, it appears to ease moderate pain (analgetic) and to be neuroprotective. THC has approximately equal affinity for the CB1 and CB2 receptors.[5] Its effects are perceived to be more cerebral.



Main classes of natural cannabinoids

delta-9-Tetrahydrocannabinol (Δ^9-THC, THC) and *delta*-8-tetrahydrocannabinol (Δ^8-THC), mimic the action of anandamide, a neurotransmitter produced naturally in the body. The THCs produce the *high* associated with cannabis by binding to the CB_1 cannabinoid receptors in the brain.

Cannabidiol

Cannabidiol (CBD) is not psychoactive, and was thought to not affect the psychoactivity of THC.[6] However, recent evidence shows that smokers of cannabis with a higher CBD/THC ratio were less likely to experience schizophrenia-like symptoms.[7] This is supported by psychological tests, in which participants experience less intense psychotic effects when intravenous THC was co-administered with CBD (as measured with a PANSS test).[8] It has been hypothesized that CBD acts as an allosteric antagonist at the CB1 receptor and thus alters the psychoactive effects of THC.

Medically, it appears to relieve convulsion, inflammation, anxiety, and nausea.[9] CBD has a greater affinity for the CB2 receptor than for the CB1 receptor.[9] It is perceived to have more effect on the body.

CBD shares a precursor with THC and is the main cannabinoid in low-THC *Cannabis* strains.

Cannabinol

Cannabinol (CBN) is the primary product of THC degradation, and there is usually little of it in a fresh plant. CBN content increases as THC degrades in storage, and with exposure to light and air. It is only mildly psychoactive. Its affinity to the CB2 receptor is higher than for the CB1 receptor.[10]

Tetrahydrocannabivarin

Tetrahydrocannabivarin (THCV) is prevalent in certain South African and Southeast Asian strains of Cannabis. It is an antagonist of THC at CB_1 receptors and attenuates the psychoactive effects of THC.[11]

Cannabichromene

Cannabichromene (CBC) is non-psychoactive and does not affect the psychoactivity of THC [6].

Double bond position

In addition, each of the compounds above may be in different forms depending on the position of the double bond in the alicyclic carbon ring. There is potential for confusion because there are different numbering systems used to describe the position of this double bond. Under the dibenzopyran numbering system widely used today, the major form of THC is called Δ^9-THC, while the minor form is called Δ^8-THC. Under the alternate terpene numbering system, these same compounds are called Δ^1-THC and Δ^6-THC, respectively.

Length

Most herbal cannabinoid compounds are 21 carbon compounds. However, some do not follow this rule, primarily because of variation in the length of the side chain attached to the aromatic ring. In THC, CBD, and CBN, this side chain is a pentyl (5 carbon) chain. In the most common homologue, the pentyl chain is replaced with a propyl (3 carbon) chain. Cannabinoids with the propyl side chain are named using the suffix "varin", and are designated, for example, THCV, CBDV, or CBNV. It appears that shorter chains increase the intensity and decrease the duration of the activity of the chemicals.

Plant profile

Cannabis plants can exhibit wide variation in the quantity and type of cannabinoids they produce. The mixture of cannabinoids produced by a plant is known as the plant's cannabinoid profile. Selective breeding has been used to control the genetics of plants and modify the cannabinoid profile. For example, strains which are used as fiber (commonly called hemp), are bred such that they are low in psychoactive chemicals like THC. Strains used in medicine are often bred for high CBD content, and strains used for recreational purposes are usually bred for high THC content, or for a specific chemical balance. Some strains of more than 20% THC in their flowering buds have been created.

Quantitative analysis of a plant's cannabinoid profile is usually determined by gas chromatography (GC), or more reliably by gas chromatography combined with mass spectrometry (GC/MS). Liquid chromatography (LC) techniques are also possible, although these are often only semi-quantitative or qualitative. There have been systematic attempts to monitor the cannabinoid profile of cannabis over time, but their accuracy is impeded by the illegal status of the plant in many countries.

Pharmacology

Cannabinoids can be administered by smoking, vaporizing, oral ingestion, transdermal patch, intravenous injection, sublingual absorption, or rectal suppository. Once in the body, most cannabinoids are metabolized in the liver, especially by cytochrome P450 mixed-function oxidases, mainly CYP 2C9. Thus supplementing with CYP 2C9 inhibitors leads to extended intoxication.

Some is also stored in fat in addition to being metabolized in liver. Δ^9-THC is metabolized to 11-hydroxy-Δ^9-THC, which is then metabolized to 9-carboxy-THC. Some cannabis metabolites can be detected in the body after several weeks.

Plant synthesis

Cannabinoid production starts when an enzyme causes geranyl pyrophosphate and olivetolic acid to combine and form CBG. Next, CBG is independently converted to either CBD or CBC by two separate synthase enzymes. CBD is then enzymatically cyclized to THC. For the propyl homologues (THCV, CBDV and CBNV), there is a similar pathway that is based on CBGV.

Separation

Cannabinoids can be separated from the plant by extraction with organic solvents. Hydrocarbons and alcohols are often used as solvents. However, these solvents are flammable and many are toxic. Supercritical solvent extraction with carbon dioxide is an alternative technique. Although this process requires high pressures (73 atmospheres or more), there is minimal risk of fire or toxicity, solvent removal is simple and efficient, and extract quality can be well-controlled. Once extracted, cannabinoid blends can be separated into individual components using wiped film vacuum distillation or other distillation techniques. However, to produce high purity cannabinoids, chemical synthesis or semisynthesis is generally required.

History

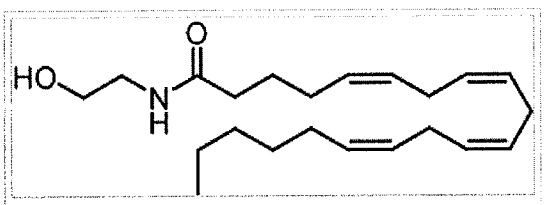
Cannabinoids were first discovered in the 1940s, when CBD and CBN were identified. The structure of THC was first determined in 1964.

Due to molecular similarity and ease of synthetic conversion, it was originally believed that CBD was a natural precursor to THC. However, it is now known that CBD and THC are produced independently in the cannabis plant.

Endocannabinoids

For more details on the roles and regulation of the endocannabinoids, see Endocannabinoid system.

Endocannabinoids are substances produced from within the body which activate cannabinoid receptors. After the discovery of the first cannabinoid receptor in 1988, scientists began searching for an endogenous ligand for the receptor.



Anandamide, an endogenous ligand of CB1 and CB2

Types of endocannabinoid ligands

- Arachidonoylethanolamine (Anandamide or AEA)

In 1992, the first such compound was identified as arachidonoyl ethanolamine and named anandamide, a name derived from the Sanskrit word for bliss and *-amide*. Anandamide is derived from the essential fatty acid arachidonic acid. It has a pharmacology similar to THC, although its chemical structure is different. Anandamide binds to the central (CB1) and, to a lesser extent, peripheral (CB2) cannabinoid receptors, where it acts as a partial agonist. Anandamide is about as potent as THC at the CB1 receptor.[12] It is found in nearly all tissues in a wide range of animals.[13]

Two analogs of anandamide, 7,10,13,16-docosatetraenoylethanolamide and *homo*-γ-linolenoylethanolamine, have similar pharmacology. All of these are members of a family of signalling lipids called *N*-acylethanolamides, which also includes the noncannabimimetic palmitoylethanolamide and oleoylethanolamine which possess anti-inflammatory and orexigenic effects, respectively. Many *N*-acylethanolamines have also been identified in plant seeds[14] and in molluscs.[15]

- 2-arachidonoyl glycerol (2-AG)

Another endocannabinoid, 2-arachidonoyl glycerol, binds to both the CB1 and CB2 receptors with similar affinity, acting as a full agonist at both.[12] 2-AG is present at significantly higher concentrations in the brain than anandamide[16], and there is some controversy over whether 2-AG rather than anandamide is chiefly responsible for endocannabinoid signalling *in vivo* [17]. In particular, one *in vitro* study suggests that 2-AG is capable of stimulating higher G-protein activation than anandamide, although the physiological implications of this finding are not yet known.[18]

- 2-arachidonyl glyceryl ether (noladin ether)

In 2001 a third, ether-type endocannabinoid, 2-arachidonyl glyceryl ether (noladin ether), was isolated from porcine brain. [19] Prior to this discovery, it had been synthesized as a stable analog of 2-AG; indeed, some controversy remains over its classification as an endocannabinoid, as another group failed to detect the substance at "any appreciable amount" in the brains of several different mammalian species.[20] It binds to the CB1 cannabinoid receptor (K_i = 21.2 nmol/L) and causes sedation, hypothermia, intestinal immobility, and mild antinociception in mice. It binds primarily to the CB1 receptor, and only weakly to the CB2 receptor.[12]

- N-arachidonoyl-dopamine (NADA)

Discovered in 2000, NADA preferentially binds to the CB1 receptor.[21] Like anandamide, NADA is also an agonist for the vanilloid receptor subtype 1 (TRPV1), a member of the vanilloid receptor family.[22][23]

- Virodhamine (OAE)

A fifth endocannabinoid, virodhamine, or *O*-arachidonoyl-ethanolamine (OAE) was discovered in June 2002. Although it is a full agonist at CB2 and a partial agonist at CB1, it behaves as a CB1 antagonist *in vivo*. In rats, virodhamine was found to be present at comparable or slightly lower concentrations than anandamide in the brain, but 2- to 9-fold higher concentrations peripherally.[24]

Function

Endocannabinoids serve as intercellular 'lipid messengers', signaling molecules that are released from one cell and activate the cannabinoid receptors present on other nearby cells. Although in this intercellular signaling role they are similar to the well-known monoamine neurotransmitters, such as acetylcholine, GABA or dopamine, endocannabinoids differ in numerous ways from them. For instance, they use retrograde signaling. Furthermore, endocannabinoids are lipophilic molecules that are not very soluble in water. They are not stored in vesicles, and exist as integral constituents of the membrane bilayers that make up cells. They are believed to be synthesized 'on-demand' rather than made and stored for later use. The mechanisms and enzymes underlying the biosynthesis of endocannabinoids remain elusive and continue to be an area of active research.

The endocannabinoid 2-AG has been found in bovine and human maternal milk.[25]

Retrograde signal

Conventional neurotransmitters are released from a 'presynaptic' cell and activate appropriate receptors on a 'postsynaptic' cell, where presynaptic and postsynaptic designate the sending and receiving sides of a synapse, respectively.

Endocannabinoids, on the other hand, are described as retrograde transmitters because they most commonly travel 'backwards' against the usual synaptic transmitter flow. They are in effect released from the postsynaptic cell and act on the presynaptic cell, where the target receptors are densely concentrated on axonal terminals in the zones from which conventional neurotransmitters are released. Activation of cannabinoid receptors temporarily reduces the amount of conventional neurotransmitter released. This endocannabinoid mediated system permits the postsynaptic cell to control its own incoming synaptic traffic. The ultimate effect on the endocannabinoid releasing cell depends on the nature of the conventional transmitter that is being controlled. For instance, when the release of the inhibitory transmitter, GABA, is reduced, the net effect is an increase in the excitability of the endocannabinoid-releasing cell. Conversely, when release of the excitatory neurotransmitter, glutamate, is reduced, the net effect is a decrease in the excitability of the endocannabinoid-releasing cell.

Range

Endocannabinoids are hydrophobic molecules. They cannot travel unaided for long distances in the aqueous medium surrounding the cells from which they are released, and therefore act locally on nearby target cells. Hence, although emanating diffusely from their source cells, they have much more restricted spheres of influence than do hormones, which can affect cells throughout the body.

Other thoughts

Endocannabinoids constitute a versatile system for affecting neuronal network properties in the nervous system.

Scientific American published an article in December 2004, entitled "The Brain's Own Marijuana" discussing the endogenous cannabinoid system. [26]

The current understanding recognizes the role that endocannabinoids play in almost every major life function in the human body.

U.S. Patent # 6630507

In 2003, the U.S. Government as represented by the Department of Health and Human Services filed for, and was awarded a patent on cannabinoids as antioxidants and neuroprotectants. U.S. Patent 6630507.

Synthetic and patented cannabinoids

Historically, laboratory synthesis of cannabinoids were often based on the structure of herbal cannabinoids and a large number of analogs have been produced and tested, especially in a group led by Roger Adams as early as 1941 and later in a group led by Raphael Mechoulam. Newer compounds are no longer related to natural cannabinoids or are based on the structure of the endogenous cannabinoids.

Synthetic cannabinoids are particularly useful in experiments to determine the relationship between the structure and activity of cannabinoid compounds, by making systematic, incremental modifications of cannabinoid molecules.

Medications containing natural or synthetic cannabinoids or cannabinoid analogs:

- Dronabinol (Marinol), is Δ9-tetrahydrocannabinol (THC), used as an appetite stimulant, anti-emetic and analgesic.
- Nabilone (Cesamet), a synthetic cannabinoid and an analog of Marinol. It is Schedule II unlike Marinol which is Schedule III.
- Sativex, a cannabinoid extract oral spray containing THC, CBD, and other cannabinoids used for neuropathic pain and spasticity in Canada and Spain. Sativex develops whole plant cannabinoid medicines.
- Rimonabant (SR141716), a selective cannabinoid (CB1) receptor antagonist used as an anti-obesity drug under the proprietary name, Acomplia. It is also used for smoking cessation.

Other notable synthetic cannabinoids include:

- CP-55940, produced in 1974, this synthetic cannabinoid receptor agonist is many times more potent than THC
- Dimethylheptylpyran
- HU-210, about 100 times as potent as THC[27].\
- HU-331 a potential anti-cancer drug derived from cannabidiol that specifically inhibits topoisomerase II.

- SR144528, a CB2 receptor antagonists
- WIN 55,212-2, a potent cannabinoid receptor agonist
- JWH-133, a potent selective CB2 receptor agonist.
- Levonantradol (Nantrodolum), an anti-emetic and analgesic but not currently in use in medicine.

Table of natural cannabinoids

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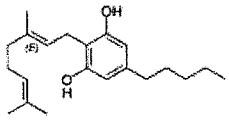
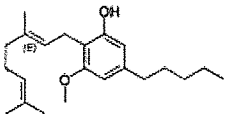
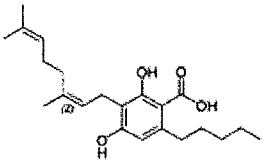
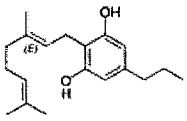
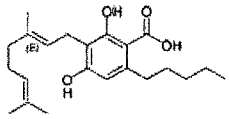
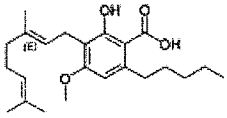
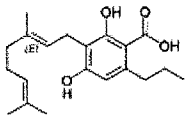
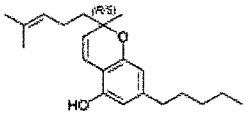
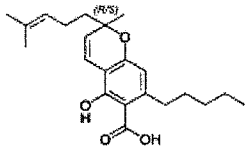
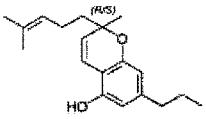
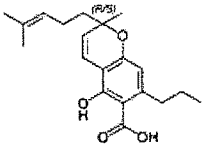
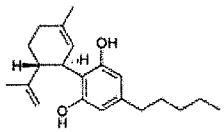
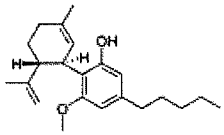
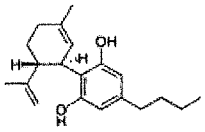
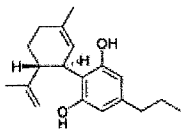
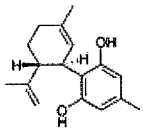
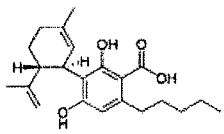
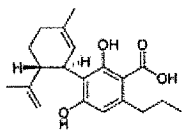
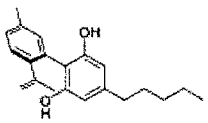
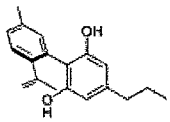
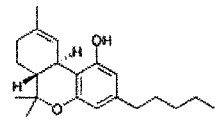
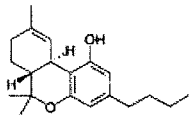
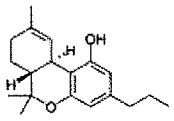
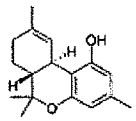
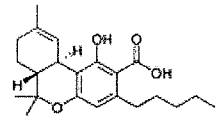
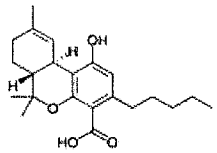
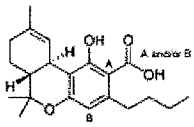
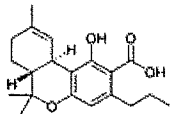
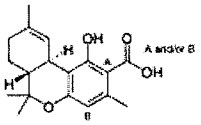
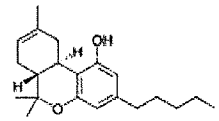
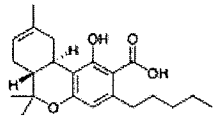
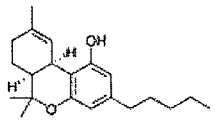
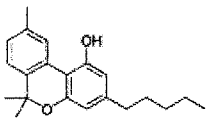
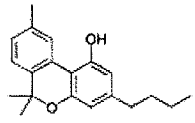
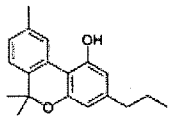
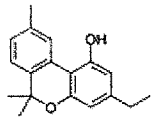
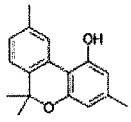
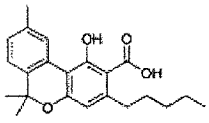
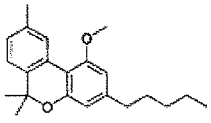
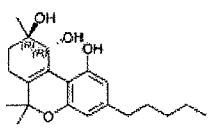
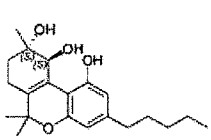
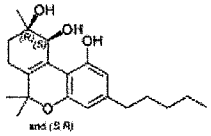
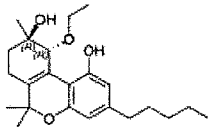
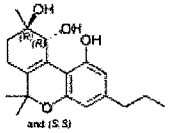
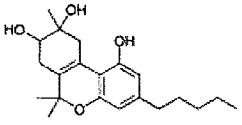
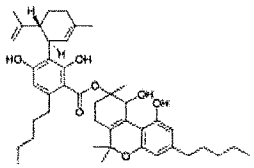
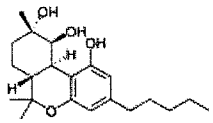
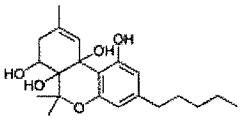
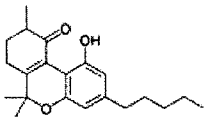
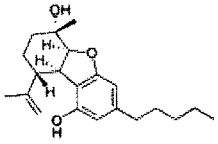
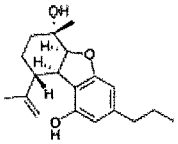
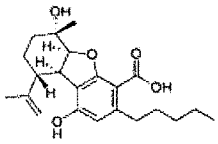
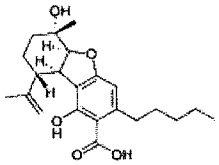
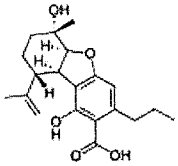
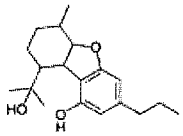
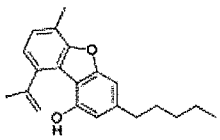
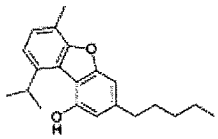
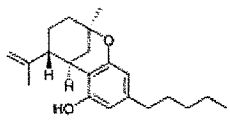
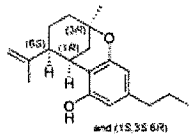
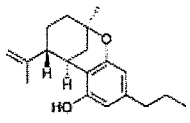
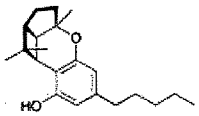
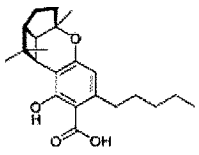
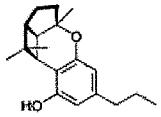
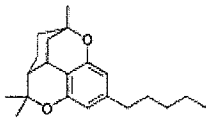
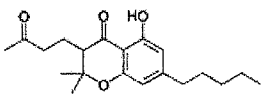
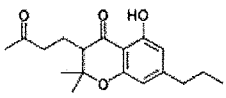
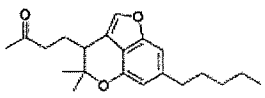
 Cannabigerol **(*E*)-CBG-C_5**	 Cannabigerol monomethyl ether **(*E*)-CBGM-C_5 A**	 Cannabinerolic acid A **(*Z*)-CBGA-C_5 A**	 Cannabigerovarin **(*E*)-CBGV-C_3**	
 Cannabigerolic acid A **(*E*)-CBGA-C_5 A**	 Cannabigerolic acid A monomethyl ether **(*E*)-CBGAM-C_5 A**		 Cannabigerovarinic acid A **(*E*)-CBGVA-C_3 A**	
Cannabichromene-type (CBC)				
 (±)-Cannabichromene **CBC-C_5**	 (±)-Cannabichromenic acid A **CBCA-C_5 A**	 (±)-Cannabivarichromene, (±)-Cannabichromevarin **CBCV-C_3**	 (±)-Cannabichromevarinic acid A **CBCVA-C_3 A**	
Cannabidiol-type (CBD)				
 (−)-Cannabidiol **CBD-C_5**	 Cannabidiol momomethyl ether **CBDM-C_5**	 Cannabidiol-C_4 **CBD-C_4**	 (−)-Cannabidivarin **CBDV-C_3**	 Cannabidiorcol **CBD-C_1**
 Cannabidiolic acid **CBDA-C_5**			 Cannabidivarinic acid **CBDVA-C_3**	
Cannabinodiol-type (CBND)				
				

Cannabinodiol **CBND-C_5**	Cannabinodivarin **CBND-C_3**			
Tetrahydrocannabinol-type (THC)				
 Δ^9-Tetrahydrocannabinol **Δ^9-THC-C_5**		 Δ^9-Tetrahydrocannabinol-C_4 **Δ^9-THC-C_4**	 Δ^9-Tetrahydrocannabivarin **Δ^9-THCV-C_3**	 Δ^9-Tetrahydrocannabiorcol **Δ^9-THCO-C_1**
 Δ^9-Tetrahydrocannabinolic acid A **Δ^9-THCA-C_5 A**	 Δ^9-Tetrahydrocannabinolic acid B **Δ^9-THCA-C_5 B**	 Δ^9-Tetrahydrocannabinolic acid-C_4 A and/or B **Δ^9-THCA-C_4 A and/or B**	 Δ^9-Tetrahydrocannabivarinic acid A **Δ^9-THCVA-C_3 A**	 Δ^9-Tetrahydrocannabiorcolic acid A and/or B **Δ^9-THCOA-C_1 An and/or B**
 (−)-Δ^8-*trans*-(6a*R*,10a*R*)-Δ^8-Tetrahydrocannabinol **Δ^8-THC-C_5**	 (−)-Δ^8-*trans*-(6a*R*,10a*R*)-Tetrahydrocannabinolic acid A **Δ^8-THCA-C_5 A**	 (−)-(6a*S*,10a*R*)-Δ^9-Tetrahydrocannabinol **(−)-*cis*-Δ^9-THC-C_5**		
Cannabinol-type (CBN)				
 Cannabinol **CBN-C_5**	 Cannabinol-C_4 **CBN-C_4**	 Cannabivarin **CBN-C_3**	 Cannabinol-C_2 **CBN-C_2**	 Cannabiorcol **CBN-C_1**
 Cannabinolic acid A **CBNA-C_5 A**	 Cannabinol methyl ether **CBNM-C_5**			
Cannabitriol-type (CBT)				
 (−)-(9*R*,10*R*)-*trans*-Cannabitriol **(−)-*trans*-CBT-C_5**	 (+)-(9*S*,10*S*)-Cannabitriol **(+)-*trans*-CBT-C_5**	 (±)-(9*R*,10*S*/9*S*,10*R*)-Cannabitriol **(±)-*cis*-CBT-C_5**	 (−)-(9*R*,10*R*)-*trans*-10-O-Ethyl-cannabitriol **(−)-*trans*-CBT-OEt-C_5**	 (±)-(9*R*,10*R*/9*S*,10*S*)-Cannabitriol-C_3 **(±)-*trans*-CBT-C_3**

 8,9-Dihydroxy-$\Delta^{6a(10a)}$-tetrahydrocannabinol **8,9-Di-OH-CBT-C_5**	 Cannabidiolic acid A cannabitriol ester **CBDA-C_5 9-OH-CBT-C_5 ester**	 (−)-(6a*R*,9*S*,10*S*,10a*R*)-9,10-Dihydroxy-hexahydrocannabinol, Cannabiripsol **Cannabiripsol-C_5**	 (−)-6a,7,10a-Trihydroxy-Δ^9-tetrahydrocannabinol **(−)-Cannabitetrol**	 10-Oxo-$\Delta^{6a(10a)}$-tetrahydrocannabinol **OTHC**
Cannabielsoin-type (CBE)				
 (5a*S*,6*S*,9*R*,9a*R*)-Cannabielsoin **CBE-C_5**		 (5a*S*,6*S*,9*R*,9a*R*)-C_3-Cannabielsoin **CBE-C_3**		
 (5a*S*,6*S*,9*R*,9a*R*)-Cannabielsoic acid A **CBEA-C_5 A**	 (5a*S*,6*S*,9*R*,9a*R*)-Cannabielsoic acid B **CBEA-C_5 B**	 (5a*S*,6*S*,9*R*,9a*R*)-C_3-Cannabielsoic acid B **CBEA-C_3 B**		
 Cannabiglendol-C_3 **OH-iso-HHCV-C_3**	 Dehydrocannabifuran **DCBF-C_5**	 Cannabifuran **CBF-C_5**		
Isocannabinoids				
 (−)-Δ^7-*trans*-(1*R*,3*R*,6*R*)-Isotetrahydrocannabinol	 (±)-Δ^7-1,2-*cis*-(1*R*,3*R*,6*S*/1*S*,3*S*,6*R*)-Isotetrahydro-cannabivarin	 (−)-Δ^7-*trans*-(1*R*,3*R*,6*R*)-Isotetrahydrocannabivarin		
Cannabicyclol-type (CBL)				
 (±)-(1a*S*,3a*R*,8b*R*,8c*R*)-Cannabicyclol **CBL-C_5**	 (±)-(1a*S*,3a*R*,8b*R*,8c*R*)-Cannabicyclolic acid A **CBLA-C_5 A**	 (±)-(1a*S*,3a*R*,8b*R*,8c*R*)-Cannabicyclovarin **CBLV-C_3**		

Cannabicitran-type (CBT)				
 Cannabicitran **CBT-C_5**				
Cannabichromanone-type (CBCN)				
 Cannabichromanone **CBCN-C_5**	 Cannabichromanone-C3 **CBCN-C_3**	 Cannabicoumaronone **CBCON-C_5**		

See also

- Cannabinoid receptor antagonist

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External links

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